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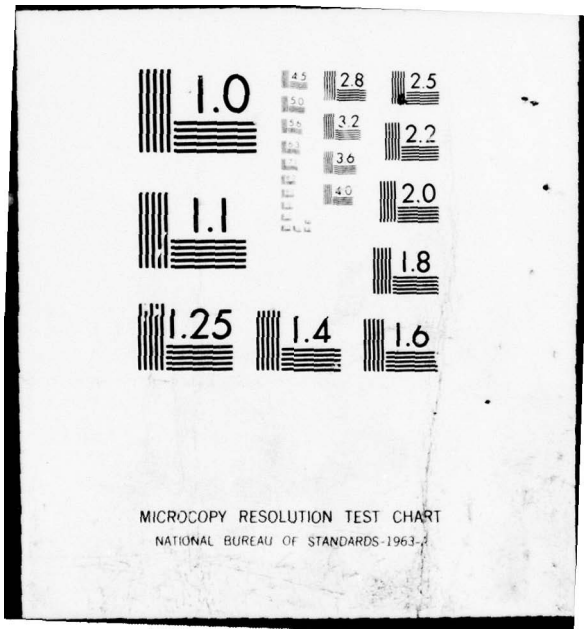
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Diving deafness and vestibular disorder can be the result of barotrauma and/or decompression sickness. Inner ear sensory hair cells are damaged with diving deafness. Also the inner ear microcirculation can be involved in the pathology. Formation of inner ear perilymph gas bubbles may also explain certain cases of diving deafness. Inner ear decompression sickness pathology is probably mediated in part through the abnormal stimulation of intravascular clotting.

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FINAL TECHNICAL REPORT OFFICE OF NAVAL RESEARCH

DIVING INDUCED DEAFNESS

Principal Investigator:

James G. McCormick, Ph.D.
Associate Professor of Otolaryngology
Director of Research Otolaryngology
Bowman Gray School of Medicine of
Wake Forest University
Winston Salem, North Carolina 27103

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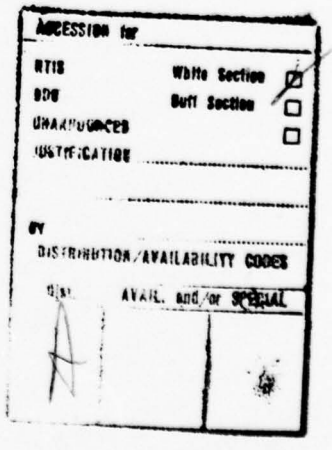

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RESEARCH COLLABORATORS

James E. Pugh, Jr., M.D.
Special NIH Research Fellow
Kresge Hearing and Research Institute
Department of Otolaryngology
University of Michigan
Medical School
Ann Arbor, Michigan

Dr. James Pugh collaborated with Dr. McCormick on the N_1N_2 eighth nerve potential study of five control squirrel monkeys and two deep hydrogen-oxygen dived squirrel monkeys with vestibular derangement. Dr. Pugh and the staff of the Kresge Hearing Research Institute at the University of Michigan Medical School made available all facilities necessary for this study including a DECK PDP12 computer.

David Anderson, Ph.D.
Assistant Professor of Otolaryngology
Kresge Hearing and Research Institute
Department of Otolaryngology
University of Michigan
Medical School
Ann Arbor, Michigan

Optokinectic electronystagmography tests on "control" and deep helium-oxygen squirrel monkeys were supervised by Dr. Anderson and carried out in his laboratory.

Ralph W. Brauer, Ph.D.
Director, Wrightsville Marine Bio-Medical Institute
University of North Carolina at Wilmington
Wilmington, North Carolina

Dr. Brauer provided us with one control squirrel monkey, one deep dived helium-oxygen squirrel monkey and one deep dived hydrogen-

oxygen squirrel monkey for N_1N_2 and electronystagmography testing at the University of Michigan.

Professor Thomas B. Clarkson
Chairman, Department of Comparative Medicine
Bowman Gray School of Medicine
Wake Forest University

Dr. Clarkson provided four control squirrel monkeys for use in N_1N_2 studies and ENG studies at the University of Michigan plus care and maintenance of animals used from Dr. Brauer's lab. Dr. Clarkson is supported by an Atherosclerosis Center Grant from the NIH Heart and Lung Institute.

Ivan L. Holleman, M.D.
Associate Professor of Pathology
Bowman Gray School of Medicine
Wake Forest University

Dr. Holleman assisted Dr. McCormick in the supervision of histology laboratory technicians. Dr. Holleman's efforts were supported by Bowman Gray School of Medicine Institutional funds.

Hersey E. Miller, M.D.
Chief Resident
Section of Otolaryngology
Bowman Gray School of Medicine
Wake Forest University

Brooks E. Neff, Ph.D.
Instructor in Otolaryngology
Bowman Gray School of Medicine
Wake Forest University

Miss Linda Gooding
Audiologist
North Carolina Baptist Hospital
Winston Salem, North Carolina

The people listed above assisted Dr. McCormick in the collection of data on the human round window fistula case. The electronystagmograph tests were carried out by Dr. Hersey Miller and the audiometric testing for this past year was carried out by Dr. Neff and Ms. Linda Gooding.

Grant H. Barlow, Ph.D.
Biochemist
Abbott Drug Research Laboratories
North Chicago, Illinois

Dr. Barlow and the Abbott Labs provided us with the urokinase for this year's studies.

RESULTS OF THE SCIENTIFIC INVESTIGATION OF DIVING DEAFNESS

Electrophysiology

Diving-deafness has been associated with loss of cochlear electric potentials from sensory hair cells, histopathological degeneration of hair cells has been shown, and these effects have been reliably studied and reproduced in the laboratory with a specific air dive schedule for guinea pigs (Figure 1), (McCormick et al., 1972, 1973[a,b,c], 1974[a,b], 1975[a,b]).

Through A.C. cochlear potential studies of approximately 100 guinea pigs it has been established that the etiology of inner ear diving-deafness can involve a malfunction of the sensory hair cells of the inner ear. This premise is also supported by celloidin inner ear histology studies of 15 of these animals (30 ears). An example of the kind of electrical loss we have seen is in Figure 2.

Histopathology

The histopathology of inner ear deafness includes hemorrhage (Figure 3). Less severe cases show a proteinaceous exudate (Figure 4) (McCormick et al., 1973, 1975). The post-dive post-decompression sickness time course of this progression of events is not completely worked out yet. Dr. McCormick's findings have been confirmed by the research of Dr. Kenneth Money, Ontario, Canada.

In a guinea pig with a diving-deafness-vestibular dysfunction after a deep helium oxygen dive and verified post-dive cochlear potential loss, hemorrhage was found bilaterally in the animal's inner ear. This hemorrhage was also associated with the occurrence of general decompression sickness without barotrauma to the middle ear systems of the animal. This finding is in keeping with the speculation of Dr. Dr. Donald Harris (Groton Submarine Medical Center, 1969) that human divers have suffered hearing loss and vestibular dysfunction from inner ear hemorrhage resulting from diving conditions. Main points of interest in the Bowman Gray guinea pig hemorrhage case were:

a. Hemorrhage affected the inner ear only at the level of the capillary bed.

b. The sensory hair cells of the cochlea were degenerated.

c. Primary vascular damage appeared to be in the capillaries under the basilar membrane, whereas the stria vascularis was only slightly damaged.

d. Hemorrhage occurred in the perilymph of the vestibular system as well as the cochlea.

Considerable evidence has been accumulated supporting the idea that hemorrhage in diving-deafness does not occur immediately post-dive. In fact it is probable that there may be several hours post-dive before hemorrhage occurs and in which time appropriate therapy might be instituted to prevent eventual inner ear hemorrhage and permanent hearing loss. This tenet is supported by the following evidence:

a. In a guinea pig decompression sickness-deafness experiment at 22 hours post-dive a proteinaceous exudate was found in the inner ears of the animal but no frank hemorrhage was apparent In vascular systems in general this is a degenerative process which takes place before further vascular wall breakdown and hemorrhage.

b. In seven post-dive cochlear potential hearing loss guinea pig cases the administration of the fibrinolytic agent urokinase did not aggravate the cochlear potential loss, and in some of these cases the administration of urokinase was associated with a leveling off of the loss or a recovery trend.

c. In five additional cases of documented cochlear potential hearing loss after the induction of decompression sickness in guinea pigs, the administration of a combination of heparin and urokinase did not result in an immediate aggravation of the

cochlear potential loss. Instead several dramatic recovery trends were noted in the first three to six hours post-dive.

Drug Treatments

In cochlear potential studies, heparin has shown a partial protective effect against decompression sickness induced deafness. Treatment drugs for diving deafness have not yet been found, but the drug urokinase shows some promise and needs further testing. (McCormick et al., 1973[a,b], 1975).

With decompression sickness induced diving deafness (A.C. cochlear potential sensitivity) animals pretreated with clinical doses of heparin had significantly less (0.05 level) cochlear potential loss post-dive than non drug treated animals. Non-parametric statistics were used for this analysis.

Extensive pre- and post-dive cochlear potential studies have been carried out on guinea pigs treated in different ways. Post-dive cochlear potential sensitivity and maximum output data was taken for all of these animals from 2 to 22 hours. Twelve control dived animals have been studied with no drug treatment. Five dived animals have been studied with post-dive urokinase treatment. Seven animals have been dived with post-dive combination of urokinase and heparin treatment and 10 animals have been dived and studied with pre-dive heparin treatment. Concerning the maximum output cochlear potential data (indicative of the dynamic

range of loudness appreciation) the following statistical evaluations have been noted:

a. Using non-parametric statistics the group of animals treated post-dive with a combination of urokinase and heparin had a significantly higher cochlear potential recovery than the contr group or the group treated post-dive with urokinase alone. Thi observation was for maximum cochlear potential data at 1,000 Hz For 10KHz. the group of animals treated with the post-dive combination of urokinase and heparin had a significantly higher recovery rate than all of the other three animal categories involved. The significance level for both 1,000 Hz. and 10KHz. was 0.05.

b. The net loss of cochlear potential maximum at 1,000 Hz. according to an analysis of variance was significantly higher at the 10% level for the post-dive urokinase group as compared to the post-dive urokinase plus heparin group and the pre-dive heparin group. Also in an analysis of variance for the net loss at the maximum recovery point for 10,000 Hz. the post-dive urokinase group was significantly greater at the 5% level than the group treated with pre-dive heparin.

c. For 1,000 Hz. maximum cochlear potential data the group of animals treated pre-dive with heparin had a significantly lower amount of loss at the 10% level than the group treated post-dive with urokinase alone. Likewise for 10KHz, the amount of cochlear potential loss for the pre-dive heparin treated group was significantly less at the 5% level than the group treated post-dive with urokinase.

d. For an analysis of variance between all groups with regard to the final amount of loss recorded at the end of the post-dive session, the group treated post-dive with urokinase had a significantly greater cochlear potential loss at 1,000 Hz. when compared with the group treated pre-dive with heparin. For 10KHz. the post-dive urokinase group also had a greater final loss than the group of animals treated pre-dive with heparin (significance at the 5% level).

Although it was noted above that post-dive treatment with a combination of urokinase and heparin resulted in a greater recovery of cochlear potential function when compared with both control groups and a group treated post-dive with urokinase alone, it should be noted that it was determined in our study that the fibrinolytic activity of the urokinase used in combination with the heparin was greater than the fibrinolytic activity of the urokinase used alone. Therefore it is not clear whether the combination of heparin and urokinase is more effective per say or whether it was just a case of the use of more effective urokinase and not the addition of the heparin to the regimen. This fact may also explain other statistically significant differences noted above between the post-dive heparin and urokinase combination group and urokinase alone. Further studies with the improved urokinase used alone post-dive are obviously called for in the future.

Inner Ear Gas Bubbles

Gas bubbles have been discovered in the perilymph of the inner ear associated with decompression sickness in the guinea pig (Figure 5) (McCormick et al., 1975). Exactly when these bubbles form during a dive and how they affect inner ear blood flow and electric function is not known.

A 300-foot air dive decompression schedule which was found to be capable of reliably producing decompression sickness and concurrent cochlear potential hearing loss in guinea pigs was also found to

produce bubbles in the perilymph of the inner ear. The inner ear fluids were examined post-dive in a pilot study of 5 guinea pigs (10 ears). Using Fisher's exact test it was found that the incidence of bubbles in the inner ear fluid of dived animals was significantly higher than control animals (no cases of bubbles) at the 0.02 level.

Subsequently, a total of 37 guinea pigs were dived on our standard 300-foot air dive and studied post-dive in a fresh condition for inner ear gas bubbles. Twenty-two of these animals were dived alive and twenty-three guinea pigs were dived dead (eight of these dived ones had previously been dived alive and no bubbles found).

The general procedure was to dive the animals anesthetized pre-dive with urethane 10 cc/kg. All animals were in the weight range of 300 to 400 grams. Some of the animals had their middle ear cavities opened pre-dive to prevent barotrauma and others were not operated pre-dive. Post-dive, the round windows of the inner ears of the animals were surgically exposed and observed with a Zeiss operating microscope, looking for perilymph bubbles in a post-dive period from 15 minutes to two hours.

We reconfirmed the presence of perilymph bubbles seen post-dive through the round window membrane in five animals examined post-dive in the fresh condition. Three of these animals (bubbles seen in one ear of each) were dived alive and two of these animals were dived dead (very small bubbles seen in both inner ears of each animal). The incidence of post-dive bubbles did not seem to be related to the presence or absence of middle ear barotrauma during the dive.

The presence of gas bubbles behind the round window of two animals dived dead implies that an intact blood circulation between the lungs and the inner ear is not necessary for the gas buildup in the inner ear fluids. However, the bubbles seen in these animals were smaller and more difficult to identify than the ones found in animals dived alive.

Audiometric and Electronystagmography Study Of Diving Induced Round Window Fistula In A Human Scuba Diver

Significant, and often permanent, hearing loss and vestibular dysfunction occur in association with rupture of the round window membrane. Three cases of round window membrane rupture in divers have previously been reported in the literature. A fourth case is presented here, along with audiologic and electronystagmographic evaluations for one year following operative repair. Significant audiovestibular improvement was observed.

It is suggested that surgical intervention should be considered in any diver who is not likely to have decompression sickness but who develops sensorineural hearing loss, or vertigo, or both following a dive.

Sensorineural hearing loss has been reported in experienced divers using standard decompression tables and may occur whether or not the diver had difficulty clearing his ears during the dive.[3,4,5,20,21] This sensorineural hearing loss may progress to total and permanent deafness. Vertigo may be present. The terminology "inner ear barotrauma" has been proposed to differentiate such cases of sensorineural hearing loss from other forms of aural barotrauma and from involvement of the eighth nerve in decompression sickness.[3]

Round window membrane ruptures have also been reported in

association with diving, Pullen[20] citing one case occurring in a scuba diver, and Freeman et al.[4] adding two others occurring in professional divers. Two of the three patients had a profound sensorineural hearing loss preceded, in one, by severe vertigo; the third had severe vestibular disturbance but no hearing loss. Round window ruptures not related to diving have also been associated with major audiovestibular abnormalities.[5,6]

Reported here is a case of round window membrane rupture occurring during a scuba dive, with audiologic and electronystagmographic (ENG) evaluation during one year's followup.

On June 8, 1974, a 25 year-old woman was scuba diving in a rock quarry at a depth of approximately 30 feet. The air in her tank became exhausted and she surfaced quickly. On reaching the shore she noticed that both her ears felt full and that she was mildly dizzy when she stood up. Later that day she developed severe vertigo, which, on the following day worsened and was accompanied by vomiting. She was first seen by a physician on June 9, and was treated with antiemetics and bed rest. At that time, otologic evaluation revealed a right serous otitis, a question of nystagmus on left lateral gaze, and a mild conductive hearing loss. The patient had poor balance, tending to fall forward. On June 12, her vertigo was much improved, but she observed tinnitus and marked diminution in the hearing in her right ear, and had difficulty using the telephone. An audiogram showed severe sensorineural hearing loss in her right ear, normal hearing in her left ear (Fig. 1). She was

immediately admitted to the hospital where her right middle ear was explored through an elevated tympanomeatal flap. This exposure revealed a small peripheral opening in the round window membrane which allowed active loss of perilymph. Closure of the opening with pledgets of absorbable gelatin sponge (Gelfoam^{R}) placed in the round window niche controlled the leakage of perilymph. On the following day the patient observed that the tinnitus had lessened and that her hearing had improved. ENG evaluation [1] done June 17 showed spontaneous nystagmus to the left only, with eyes closed, and positional nystagmus in the same direction (Figure 6). Audiometric findings are shown in Figure 7-13. Vestibular and audiologic studies done at intervals over the next year are shown in Table 1 and Figures 3 through 7.

Although Simmons[21] in 1968 proposed inner ear membrane breaks as a cause of audiovestibular abnormalities, Goodhill[5] in 1971 was apparently the first to report round or oval window fistulae in association with sudden hearing loss. Pullen[20] soon reported four cases of round window fistulae and Goodhill et al.[6] an additional 15 cases of either oval or round window fistulae.

In 1972, Freeman and Edmonds[3] discussed sensorineural hearing loss from aural barotrauma after shallow water dives,* but a possible mechanism other than decompression sickness was not demonstrated until Pullen,[20] later that year, explored the ear of a diver sustaining such a hearing loss after a dive and discovered a round window fistula. Pullen's patient had no vestibular disturbances and had complete auditory

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*To a depth no greater than 33 feet (1 atmosphere)[1]

recovery postoperatively. In 1974, Freeman et al.[4] undertook surgical exploration of the middle ear in two similar cases, finding round window rupture in both patients. Although prompt audiovestibular improvement was observed after the fistulae were closed, ENG abnormalities were still seen two months later in one of the two patients. In the present case, a persistent high frequency hearing loss could be seen post-operatively, as well as a direction changing positional nystagmus in the presence of normal caloric responses. Since no audiogram done before the injury was available, we cannot determine when this persistent hearing loss occurred. The positional nystagmus suggests, despite normal caloric tests, that the vestibular apparatus has been significantly injured, and that compensation by the vestibular nuclei has occurred but is inadequate.[7]

In all four of these diving cases, significant improvement in audiologic or audiovestibular function was observed postoperatively, and was believed to be due to the surgical intervention. In the non-diving cases reported by Goodhill et al.,[6] significant postoperative hearing gain was also noted in patients having perilymph fistulae, as compared to those in whom no fistula was demonstrated.

From these findings it can be seen that even shallow-water divers are at risk to sustain oval or round window ruptures, and consideration of this possibility should be given in divers sustaining audiovestibular injuries not believed to be due to decompression sickness. Although vestibular symptoms, when present, appear to start early, sensorineural hearing loss may not develop for hours to days. Surgical intervention should be considered when perilymph fistulae are suspected.

Dived Survival Studies of Vestibular and Auditory Function

Heretofore all of our guinea pig 300-ft. air dive experiments have been on acute non-recovery anesthetized animals. Thus -- although aware of 15 to 80 db post-dive cochlear potential losses -- we have not made any assessments of the post-dive vestibular function of our animals for the long term changes in inner ear electrophysiology or histopathology. One reason for the acute work has been the pre-dive surgery needed for electric recording from the cochlea. For these past studies we used urethane, a relatively long acting anesthetic with little or no recovery prognosis.

To assess long term electrical, and pathological conditions of our animals as well as post-dive vestibular function we dived a series of eight guinea pigs on our standard 300-ft. dive schedule with no pre-dive surgery or cochlear potential measurements and nembutal anesthesia instead of urethane (anesthesia is needed to prevent post-dive death from convulsions and possible respiratory paralysis induced by decompression sickness).

Post-dive when the animals recovered from the anesthetic, as well as 24 hours post-dive, there were no signs of any vestibular dysfunction in any of the eight guinea pigs tested, i.e., no spontaneous nystagmus was present on gross observation and the animals could walk and run in a normal manner. Pryor auditory reflexes were also normal. This is a marked contrast to the total lack of motor coordination seen post-dive in our deep helium-oxygen bends guinea pig case with inner ear hemorrhage reported in previous years and publications.

Two weeks post-dive we ran complete bilateral cochlear potential sensitivity and input output functions for each animal with subsequent perfusion and sacrifice for inner ear histology. Without pre-dive cochlear potential results for comparison it is difficult to say for sure whether there was a two week post-dive loss in inner ear electrical function; however, it was clear that any loss if present at all by two weeks was very moderate. The sensitivity in input output function of the animals were within the limits that we routinely find for non-dived animals in our laboratory. Again this is a marked contrast to our helium-oxygen bends animal which had almost total loss of inner ear electric function measured two weeks post dive.

It is possible that a recovery process took place in these ears. It is also possible that the different anesthetic nembutal may have been an important variable in the study. In our last report we postulated that a stress reaction in the autonomic nervous system might be involved in diving deafness syndrome. Perhaps nembutal is a better blocker of the autonomic nervous system than urethane, our usual agent (the helium-oxygen case was dived with no anesthetic). This point must await further investigation.

N_1N_2 and Electronystagmography Tests of Deep Dived Squirrel Monkeys

NOTE: As per the collaborator acknowledgment section of this report these studies were carried out at the Kresge Hearing Research Institute in the University of Michigan Medical Center. Dr. James Pugh was the key collaborator for the N_1N_2 studies and Dr. David Anderson was the main collaborator for the vestibular studies.

Three deep-dived (two hydrogen-oxygen, one helium-oxygen) squirrel monkeys with post-dive bends after dives at the Wrightsville Marine Bio-Medical Institute (43 D, 27 D, and 221 D) have demonstrated normal N_1N_2 auditory function and abnormal vestibular function as indicated by cold water calorics and rotatory electronystagmography tests.

Last year we reported on cold water caloric tests for squirrel monkeys 43 D and 27 D (left - no response, right - no response) and 221 D (left - O.K., right - no response). We also reported normal N_1N_2 auditory function for 27 D. This year we found normal N_1N_2 function for 27 D and 43 D (as compared to last year's controls) and grossly abnormal post-rotatory electronystagmography results for 43 D. Base-line control and post-rotatory nystagmus data was also obtained this year for three normal squirrel monkeys all of whom gave identical results.

As per Figures 8 and 9 the animals were placed in a restraining chair on a rotational platform. A head-holder kept the animal's head down at a 45° angle and motionless with a respect to the head-holder

and platform. Three pick-up flat metal plate electrodes were placed over the left and right eyes with a third indifferent in the middle of the forehead. No drugs or anesthetics were given to the animal.

The electrode signal was picked up by a Grass P18 microelectrode DC amplifier with rise time set at 100 micro seconds and an amplification factor of 100X. A 3960 Hewlett Packard instrumentation recorder was used to store data along with display of the signal on a Tektronix type RM 565 dual-beam oscilloscope. The oscilloscope output was sampled by a PDP 12 digital computer and displayed real time on the computer output screen.

Each animal was tested for optokinectic nystagmus and post-rotatory nystagmus for 15 seconds of 30 RPM and 50 RPM rotation in the clockwise and counterclockwise direction. All animals showed optokinectic nystagmus. An abnormal response was total lack of post-rotatory nystagmus. During the test the animal's eyes were covered with blinders.

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FIGURES

Figure 1:

DIVING CHART
PRNC-DSDS-7 (REV. 5-61)

NAME OF DIVER: Guinea Pig #301		RATE	TABLE USED: 300/15	DATE: July 7, 1971	
NAME OF DIVER		RATE	TENDER (Sign Name)		
PURPOSE OF DIVE			INSTRUCTOR		
LEFT SURFACE: 3:06	REACHED BOTTOM: 3:15	DESCENT TIME: 9 mins.	LEFT BOTTOM: 3:21	TIME TO FIRST STOP	TOTAL BOTTOM TIME: 15
DEPTH IN FEET	TOTAL DECOMPRESSION TIME: 15::55		TOTAL TIME OF DIVE: 30::55		PRESSURE IN POUNDS

DIVE RECORD			DEPTH OF STOP	LBS. PRESSURE	TIME
			130	58	REACHED / LEFT
			120	54	REACHED / LEFT
			110	49	REACHED / LEFT
			100	44.5	REACHED / LEFT
9:00			90	40	REACHED / LEFT
			80	36	REACHED / LEFT
			70	32	REACHED / LEFT
		4::15	60	27	REACHED / LEFT
		10	50 2	22	REACHED 3:25::15 / LEFT 3:27::15
		10	40 3	18	REACHED 3:27::25 / LEFT 3:30::25
		10	30 6	13	REACHED 3:30::35 / LEFT 3:36::35
			20 skip this ~~15~~	9	REACHED / LEFT
		20	10 skip this ~~20~~	4.5	REACHED / LEFT

REACHED SURFACE: 3:36::55	DIVER'S CONDITION
NEW GROUP	REMARKS (Continue on reverse if necessary) Ascending a foot/second or 60 feet/minute

Air dive schedule for dive-deafness studies on guinea pigs. Used for previous electrophysiological and histopathological studies and proposed also for initial inner ear filming experiments.

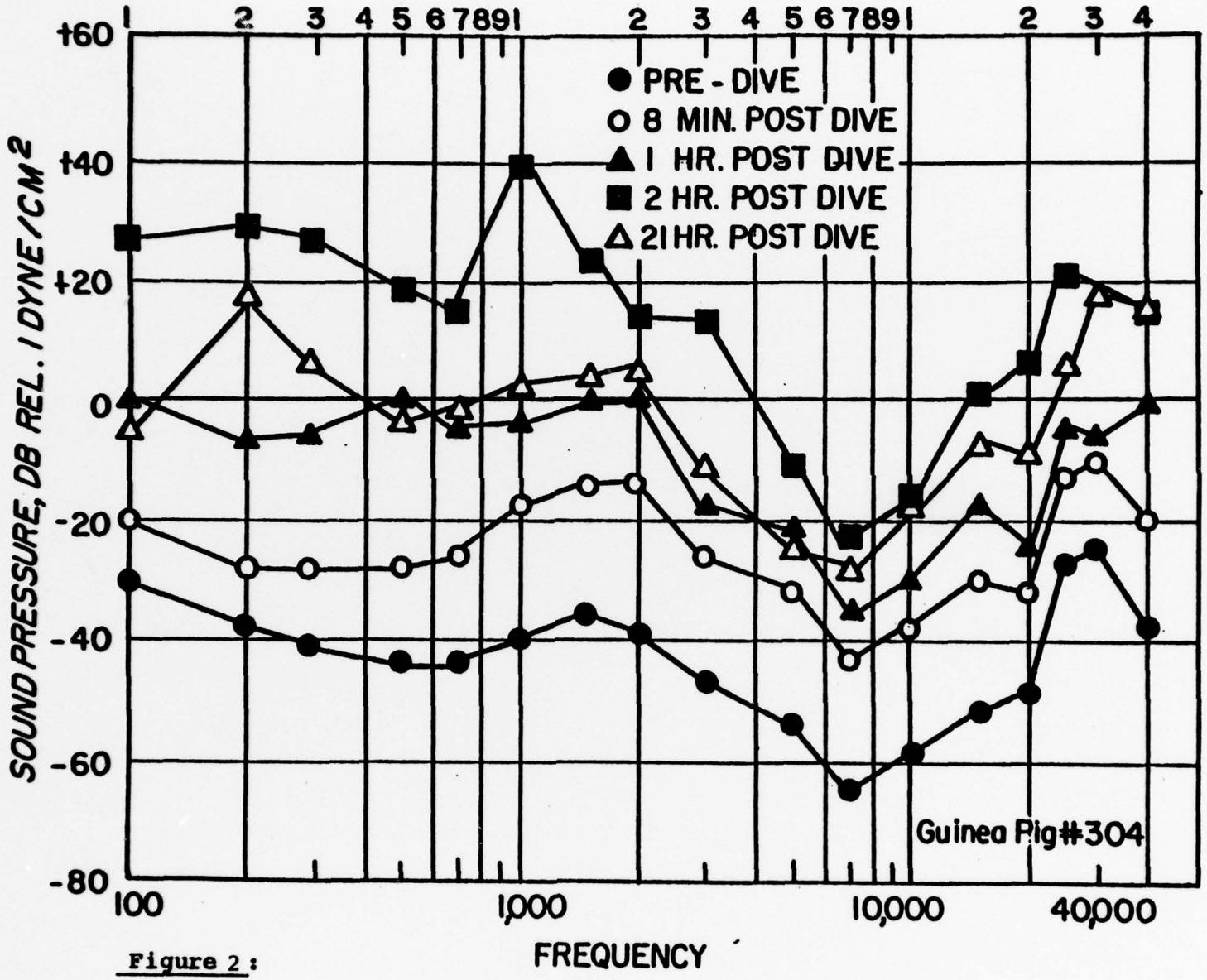


Figure 2:

Post-dive A.C. cochlear potentials for 300 foot air dived animal with proteinaceous inner ear exudate.

FIGURE 3:

Inner ear hemorrhage in a mid-modiolar section of the WMBI $He-O_2$ guinea pig with the post-dive deafness, vestibular attack, and cochlear potential loss. Fixed 14 days Post-dive, 200X.

FIGURE 4:

Proteinaceous exudate in a mid-modiolar section of the inner ear of an animal dived on the 300-foot air profile. Sacrifice 22 hours post-dive, 200X.

FIGURE 5:

Two bubbles just inside the round window membrane of a guinea pig exposed to our standard 300 foot air dive decompression profile. The manubrium of the eardrum shows in the left hand side of the print. Magnification = 50X.

Figure 6:

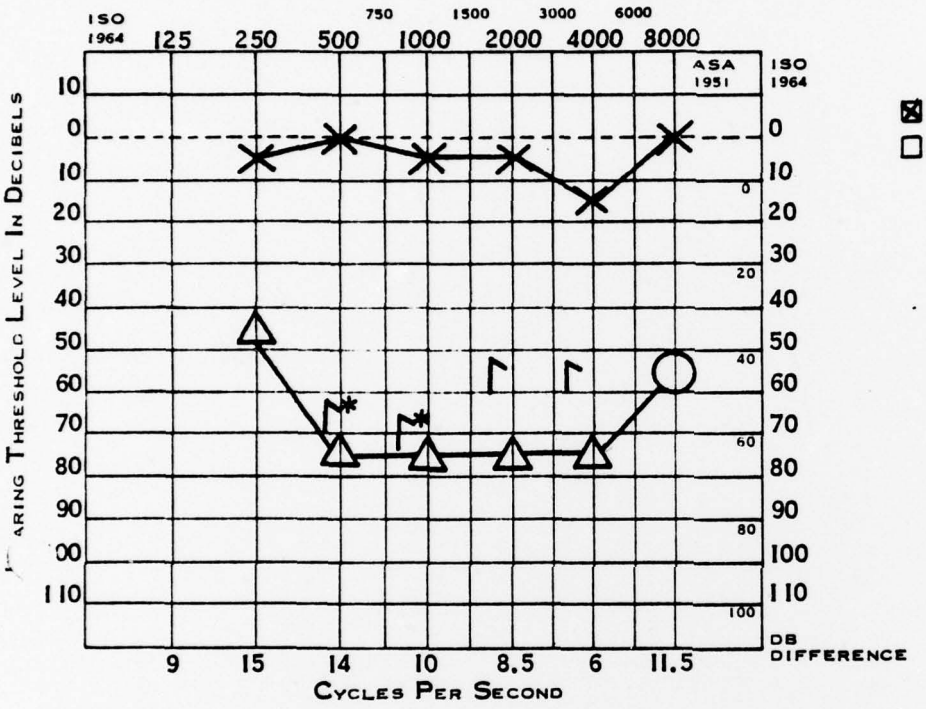
ELECTRONYSTAGMOGRAPHY RESULTS

Date		June 17, 1974	July 25, 1974	Dec. 4, 1974	May 23, 1975
Spontaneous nystagmus (eyes closed, sitting)		Moderate intensity to the left	None	None	None
Gaze Test		No nystagmus	No nystagmus	No nystagmus	No nystagmus
Optokinetic Test		Normal	Normal	Normal	Normal
Position Tests	Supine	Moderate intensity to the left	Low intensity to the right	Intermittent bidirectional, R>L	Intermittent bidirectional, R>L
	Head Left	Intense nystagmus to the left	Intense nystagmus to the right	Intense nystagmus to the left	Intense nystagmus to the left
	Head Right	Moderate intensity to the left	Intense nystagmus to the left	Intense nystagmus to the left	Intense nystagmus to the right
	Head Hanging	Low-moderate intensity, to left	Intense nystagmus to the left	Intense nystagmus to the left	Moderate intensity nystagmus to the left
Hallpike Test		To right=right-beating nyst. To left=left-beating nyst.	To right=left-beating nyst. To left=right-beating nyst.	To right=left-beating nyst. To left=right-beating nyst.	To right=left-beating nyst. To left=right-beating nyst.
Caloric Test		Spontaneous nyst. reversed with left warm irrigation (right not done)	Borderline unilateral weakness, right	Unilateral weakness > 20% right	Unilateral weakness > 20% right

Figure 7:

Pre-operative audiogram four days post-dive. Impression: Normal sensitivity in left ear and severe, essentially sensori-neural threshold shift in right ear.

PURE TONE AUDIOMETRY



STANDARD USED

☒ 1964 ISO REFERENCE THRESHOLDS

☐ 1951 ASA REFERENCE THRESHOLDS

LEGEND

	R	L
AIR CONDUCTION	O	x
MASKED	△	▽
BONE CONDUCTION	[	]
MASKED	⊢	⊣
A. C. NOT HEARD	♀	♂
B. C. NOT HEARD	⇂	⇃

MASKING

	R	L
OPPOS. EAR MASKED		
AIR CONDUCTION	db	db
BONE CONDUCTION	db	db

TYPE OF MASKING:

RELIABILITY

GOOD X FAIR ____ POOR ____

SPEECH AUDIOMETRY

EAR	SPEECH RECEPT. THRESH	SPEECH DISCRIM.	OTHER	TOLERANCE	MASKING LEVEL IN OPPS. EAR
RIGHT	db	%			db
LEFT	db	%			db
FREE FIELD	db	%			

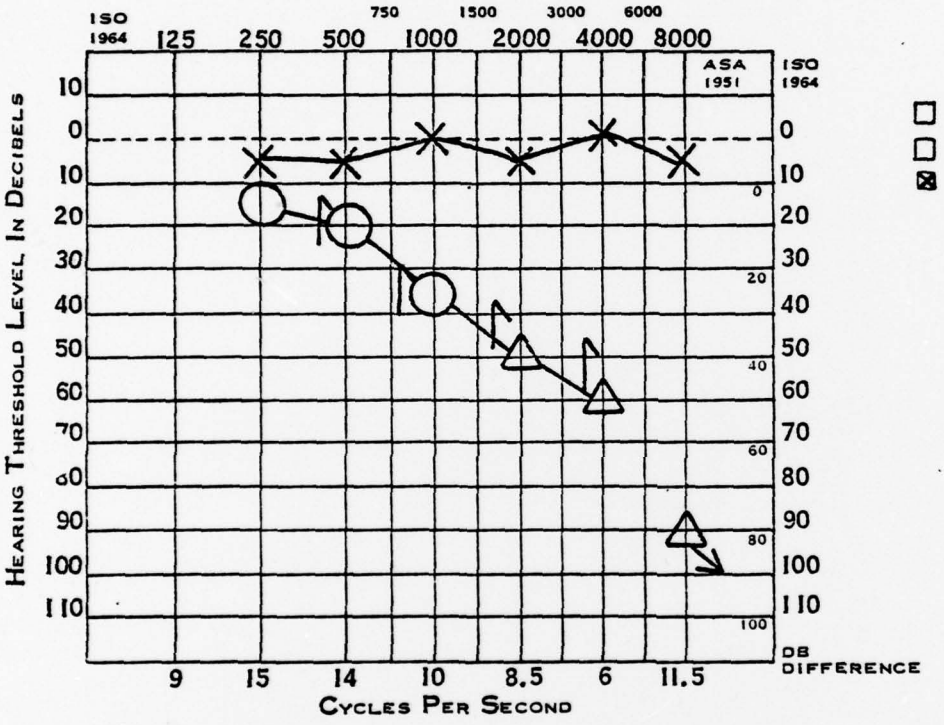
SPECIAL TESTS

*Patient noted these responses were to vibrotactile stimulation.

Figure 8:

First post-operative audiogram. Right ear was operated for round window fistula 6/12/74. Six days post-operative surgical packing still in right ear. Impression: Normal Hearing and discrimination ability in left ear. Moderate, sensori-neural threshold shift in higher frequencies, right ear. Fair speech discrimination in right ear.

PURE TONE AUDIOMETRY



STANDARD USED

- ☐ 1964 ISO REFERENCE THRESHOLDS
- ☐ 1951 ASA REFERENCE THRESHOLDS
- ☒ 1969 ANSI

LEGEND

	R	L
AIR CONDUCTION	O	X
MASKED	△	▽
BONE CONDUCTION	[	]
MASKED	⊓	⊔
A. C. NOT HEARD	O↓	X↓
B. C. NOT HEARD	⊓↓	⊔↓

MASKING

	R	L
OPPOS. EAR MASKED		
AIR CONDUCTION	db	db
BONE CONDUCTION	db	db

TYPE OF MASKING:

RELIABILITY

GOOD X FAIR ____ POOR ____

SPEECH AUDIOMETRY

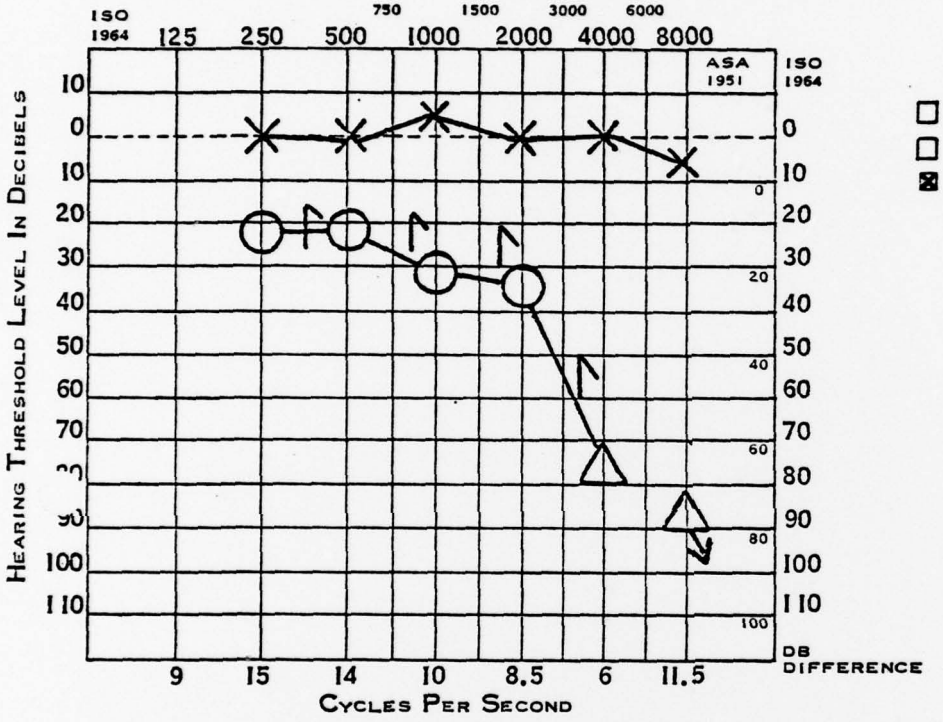
EAR	SPEECH RECEPT. THRESH	SPEECH DISCRIM.	OTHER	TOLERANCE	MASKING LEVEL IN OPPS. EAR
RIGHT	30 db	72 %	@70db		60 db
LEFT	5 db	96 %	@45db		db
FREE FIELD	db	%			

SPECIAL TESTS

Figure 9:

Second post-operative audiogram. Right Ear was operated for Round Window Fistula 6/12/74. Nine days post-operative surgical packing removed from right ear. Impression: No significant difference in thresholds (A/C or B/C) from last test date.

PURE TONE AUDIOMETRY



STANDARD USED

- ☐ 1964 ISO REFERENCE THRESHOLDS
- ☐ 1951 ASA REFERENCE THRESHOLDS
- ☒ 1969 ANSI

LEGEND

	R	L
AIR CONDUCTION	O	X
MASKED	△	▽
BONE CONDUCTION	[	]
MASKED	⊐	⊏
A. C. NOT HEARD	O↓	X↓
B. C. NOT HEARD	[↓	]↓

MASKING

	R	L
OPPOS. EAR MASKED		
AIR CONDUCTION	db	db
BONE CONDUCTION	db	db

TYPE OF MASKING:

RELIABILITY

GOOD X FAIR ____ POOR ____

SPEECH AUDIOMETRY

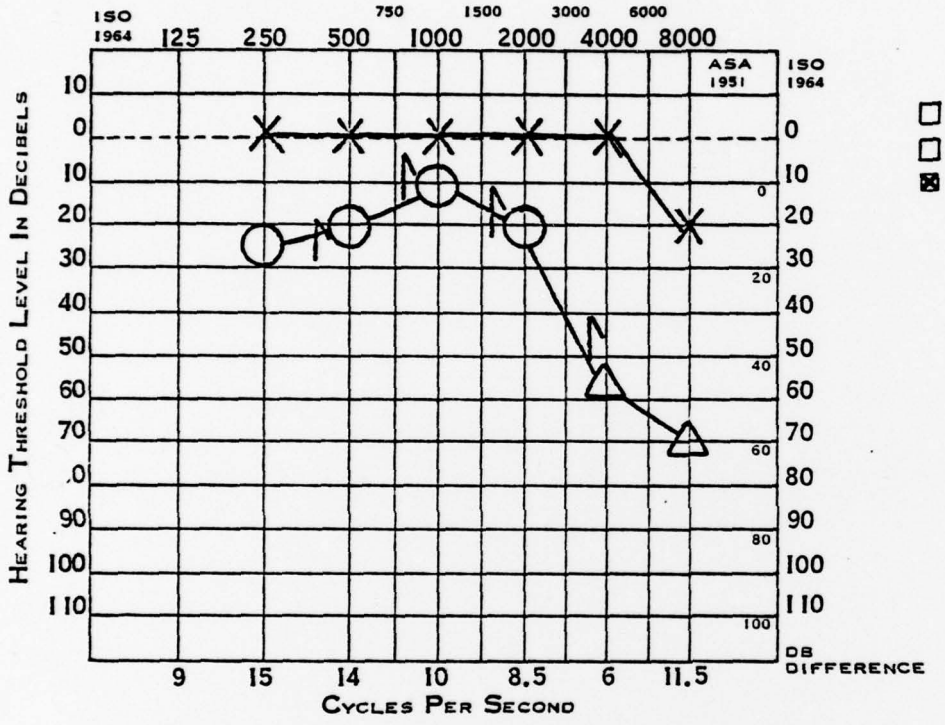
EAR	SPEECH RECEPT. THRESH	SPEECH DISCRIM.	OTHER	TOLERANCE	MASKING LEVEL IN OPPS. EAR
RIGHT	db	%			db
LEFT	db	%			db
FREE FIELD	db	%			

SPECIAL TESTS

Figure 10:

Third post-operative audiogram. Right Ear was operated for Round Window Fistula 6/12/74. Twenty-six days post-operative. Impression: Thresholds at right ear improved from 6/18/74.

PURE TONE AUDIOMETRY



Standard Used

- ☐ 1964 ISO REFERENCE THRESHOLDS
- ☐ 1951 ASA REFERENCE THRESHOLDS
- ☒ 1969 ANSI

Legend

	R	L
AIR CONDUCTION	O	x
MASKED	△	▽
BONE CONDUCTION	[	]
MASKED	⊢	⊣
A. C. NOT HEARD	♀	⚥
B. C. NOT HEARD	⟦	⟧

Masking

	R	L
OPPOS. EAR MASKED		
AIR CONDUCTION	db	80db
BONE CONDUCTION	db	100db

TYPE OF MASKING:

Reliability

GOOD X FAIR ____ POOR ____

SPEECH AUDIOMETRY

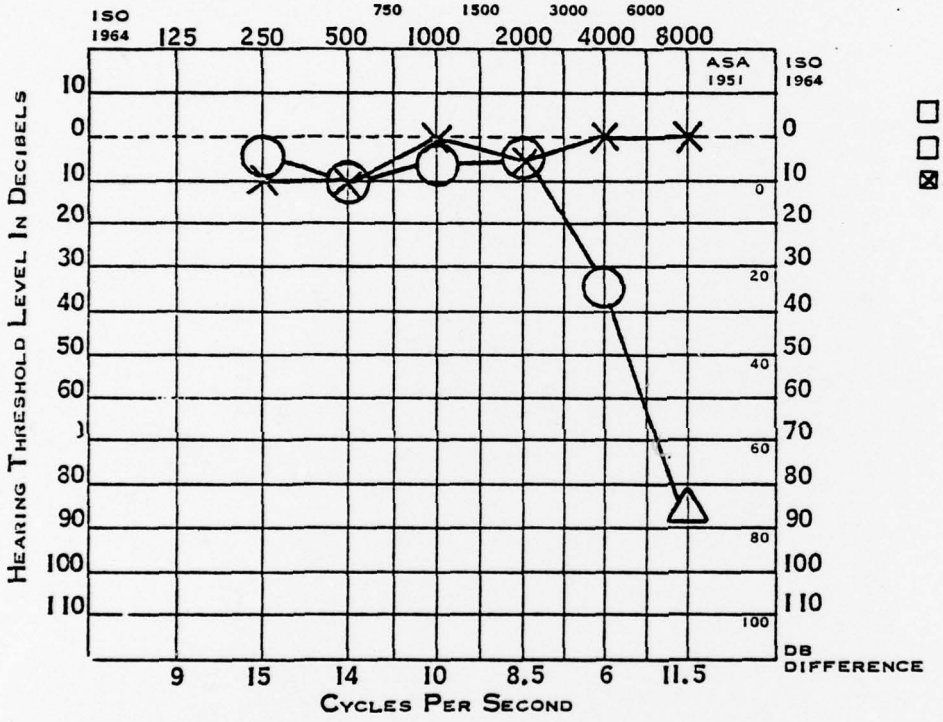
EAR	SPEECH RECEPT. THRESH	SPEECH DISCRIM.	OTHER	TOLERANCE	MASKING LEVEL IN OPPS. EAR
RIGHT	db	%			db
LEFT	db	%			db
FREE FIELD	db	%			

SPECIAL TESTS

Figure 11:

Three months, Twelve days post-operative.

PURE TONE AUDIOMETRY



Standard Used

- ☐ 1964 ISO REFERENCE THRESHOLDS
- ☐ 1951 ASA REFERENCE THRESHOLDS
- ☒ 1969 ANSI

Legend

	R	L
AIR CONDUCTION	O	x
MASKED	△	▽
BONE CONDUCTION	[	]
MASKED	⊢	⊣
A. C. NOT HEARD	♀	✱
B. C. NOT HEARD	⇩	⇩

Masking

	R	L
OPPOS. EAR MASKED		
AIR CONDUCTION	db	db
BONE CONDUCTION	db	db

TYPE OF MASKING:

Reliability

GOOD X FAIR ____ POOR ____

SPEECH AUDIOMETRY

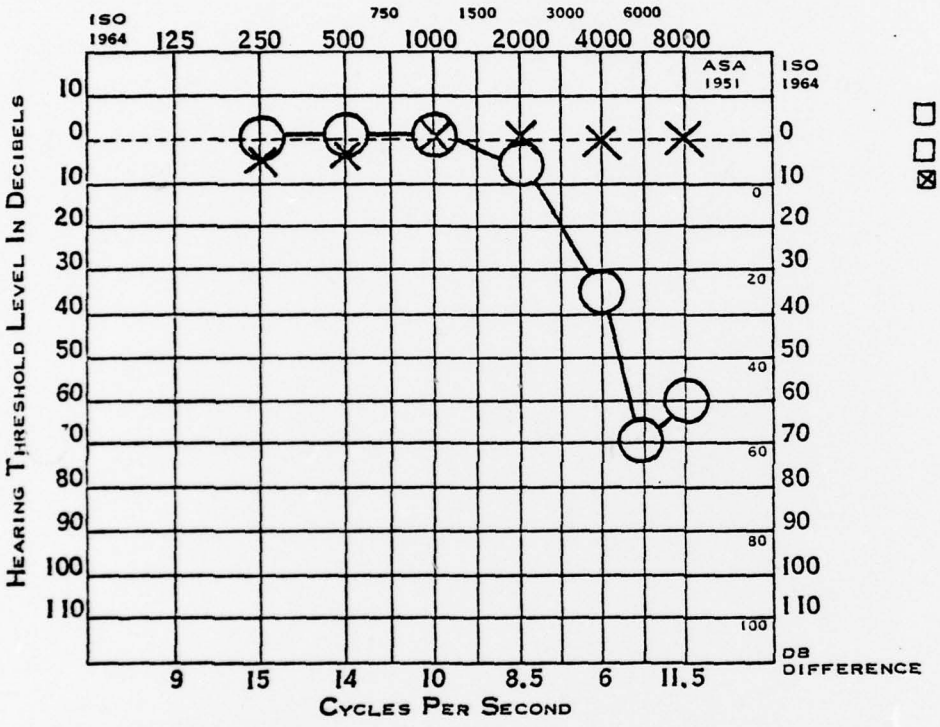
EAR	SPEECH RECEPT. THRESH	SPEECH DISCRIM.	OTHER	TOLERANCE	MASKING LEVEL IN OPPS. EAR
RIGHT	10 db	92 %	@50 db		db
LEFT	5 db	100 %	@45 db		db
FREE FIELD	db	%			

SPECIAL TESTS

Figure 12:

Six months, Four days post-operative. Impression: No significant change.

PURE TONE AUDIOMETRY



STANDARD USED

- ☐ 1964 ISO REFERENCE THRESHOLDS
- ☐ 1951 ASA REFERENCE THRESHOLDS
- ☒ 1969 ANSI

LEGEND

	R	L
AIR CONDUCTION	O	x
MASKED	△	▽
BONE CONDUCTION	[	]
MASKED	⊢	⊣
A. C. NOT HEARD	O↓	x↓
B. C. NOT HEARD	[↓	]↓

MASKING

	R	L
OPPOS. EAR MASKED		
AIR CONDUCTION	db	db
BONE CONDUCTION	db	db

TYPE OF MASKING:

RELIABILITY

GOOD OK* FAIR ____ POOR ____

SPEECH AUDIOMETRY

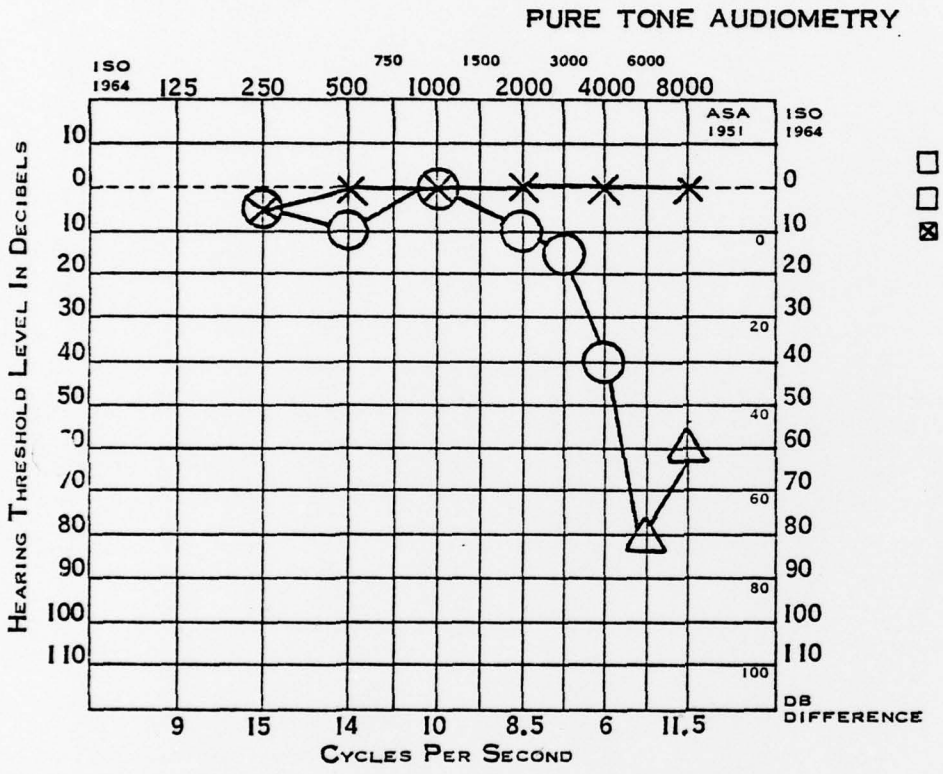
EAR	SPEECH RECEPT. THRESH	SPEECH DISCRIM.	OTHER	TOLERANCE	MASKING LEVEL IN OPPS. EAR
RIGHT	5 db	88 %	@50 db		db
LEFT	0 db	100 %	@45 db		db
FREE FIELD	db	%			

SPECIAL TESTS

*Examiner noted many false-positives. Patient noted confusion between tinnitus and test tone.

Figure 13:

Eleven months, Eleven days post-operative.



STANDARD USED

- ☐ 1964 ISO REFERENCE THRESHOLDS
- ☐ 1951 ASA REFERENCE THRESHOLDS
- ☒ 1969 ANSI

LEGEND

	R	L
AIR CONDUCTION	O	x
MASKED	△	▽
BONE CONDUCTION	[	]
MASKED	⊏	⊐
A. C. NOT HEARD	O↓	x↓
B. C. NOT HEARD	[↓	]↓

MASKING

	R	L
OPPOS. EAR MASKED		
AIR CONDUCTION	db	db
BONE CONDUCTION	db	db

TYPE OF MASKING:

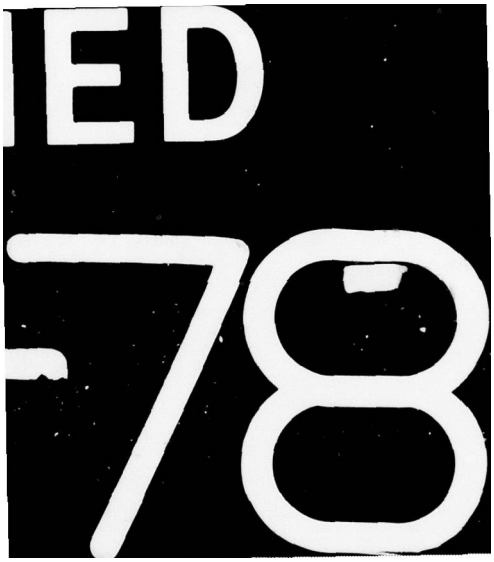
RELIABILITY

GOOD X FAIR ____ POOR ____

SPEECH AUDIOMETRY

EAR	SPEECH RECEPT. THRESH	SPEECH DISCRIM.	OTHER	TOLERANCE	MASKING LEVEL IN OPPS. EAR
RIGHT	10 db	100 %	@50 db		db
LEFT	5 db	100 %	@45 db		db
FREE FIELD	db	%			

SPECIAL TESTS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